AD-A103 368 SCHOOL OF AEROSPACE MEDICINE BROOKS AFB TX F/G 6/5
MEASUREMENT OF CORONARY-PRONE BEHAVIOR AND AUTONOMIC REACTIVITY--ETC(U)
MAY 81 C E DANIELS
UNCLASSIFIED SAM-TR-81-2 NL

1 OF 1
AD A
103368

END
DATE
FILMED
10-81
DTIC

Report SAM-TR-81-2

MEASUREMENT OF CORONARY-PRONE BEHAVIOR AND AUTONOMIC REACTIVITY

Craig E. Daniels, Ph.D.

May 1981

Final Report for Period 23 June 1980 - 29 August 1980

Approved for public release; distribution unlimited.

USAF SCHOOL OF AEROSPACE MEDICINE
Aerospace Medical Division (AFSC)
Brooks Air Force Base, Texas 78235

NOTICES

This final report was submitted by personnel of the Flight Medicine Branch, Clinical Sciences Division, USAF School of Aerospace Medicine, Aerospace Medical Division, AFSC, Brooks Air Force Base, Texas, under job order 7755-18-13.

This technical report has been reviewed and is approved for publication.

CRAIG E. DANIELS, Ph.D.
Project Scientist

GIL D. TOLAN, Lieutenant Colonel, USAF, MC
Supervisor

ROY L. DEHART
Colonel, USAF, MC
Commander

UNCLASSIFIED

SECURITY CLASSIFICATION OF THIS PAGE (When Data Entered)

REPORT DOCUMENTATION PAGE		READ INSTRUCTIONS BEFORE COMPLETING FORM
1. REPORT NUMBER 14 SAM-TR-81-2	2. GOVT ACCESSION NO. AD-A103368	3. RECIPIENT'S CATALOG NUMBER
4. TITLE (and Subtitle) 6 MEASUREMENT OF CORONARY-PRONE BEHAVIOR AND AUTONOMIC REACTIVITY		5. TYPE OF REPORT & PERIOD COVERED 9 Final Report, 23 Jun [redacted] - 29 Aug [redacted]80
		6. PERFORMING ORG. REPORT NUMBER
7. AUTHOR(s) 10 Craig E. Daniels, Ph.D.		8. CONTRACT OR GRANT NUMBER(s)
9. PERFORMING ORGANIZATION NAME AND ADDRESS USAF School of Aerospace Medicine (NGF) Aerospace Medical Division (AFSC) Brooks Air Force Base, Texas 78235		10. PROGRAM ELEMENT, PROJECT, TASK AREA & WORK UNIT NUMBERS 62202F 17 16 7755-18-13 12
11. CONTROLLING OFFICE NAME AND ADDRESS USAF School of Aerospace Medicine (NGF) Aerospace Medical Division (AFSC) Brooks Air Force Base, Texas 78235		12. REPORT DATE 11 May [redacted]81
		13. NUMBER OF PAGES 46
14. MONITORING AGENCY NAME & ADDRESS(if different from Controlling Office)		15. SECURITY CLASS. (of this report) Unclassified
		15a. DECLASSIFICATION DOWNGRADING SCHEDULE

16. DISTRIBUTION STATEMENT (of this Report)

Approved for public release; distribution unlimited.

17. DISTRIBUTION STATEMENT (of the abstract entered in Block 20, if different from Report)

18. SUPPLEMENTARY NOTES

19. KEY WORDS (Continue on reverse side if necessary and identify by block number)

Autonomic nerv. system	Cardiovascular	Blood pressure
Coronary-prone behavior	Atherosclerosis	Pulse wave velocity
Autonomic reactivity	Stress testing	Respiration
Sympathetic nerv. system	Heart rate	Electrodermal
	EMG	Salivation

20. ABSTRACT (Continue on reverse side if necessary and identify by block number)

In spite of a slight decrease in the number of fatal myocardial infarctions over the last few years, coronary disease remains this country's number one killer. A recent U.S. Air Force report estimated that cardiovascular incidents involving active duty personnel cost the Air Force $64 million per year.

In addition to traditional risk factors (e.g., age, smoking, cholesterol level), the concepts of coronary-prone behavior and stress responses as

DD FORM 1 JAN 73 1473

UNCLASSIFIED 311000

SECURITY CLASSIFICATION OF THIS PAGE (When Data Entered)

20. ABSTRACT (Continued)

major factors in the development of coronary disease have, within the last decade, received wide support. Research on the Type A coronary-prone behavior pattern has shown Type A's to be higher than Type B's on a number of biochemical and psychophysiological variables associated with accelerated development of atherosclerosis.

Current methods of assessing coronary-prone behavior using either the structured interview or the Jenkin's self-report questionnaire have inherent methodological limitations or assumptions that substantially reduce their utility, especially when they are used to help detect subclinical heart disease in an educated population, such as Air Force pilots, which is highly motivated not to display cardiovascular disease symptomatology.

Since hyperreactivity or hyperlability of the sympathetic nervous system is thought to be the major physiological mechanism underlying coronary-prone behavior, an extensive review of the literature was undertaken to determine whether it would be feasible to measure coronary-prone behavior more directly by means of autonomic response profiles. On the basis of this review, I consider such a project to be feasible and recommend a multistage project for the development and testing of a protocol to measure autonomic reactivity under stressful conditions.

ACKNOWLEDGMENT

I would like to thank the Air Force Systems Command, the Air Force Office of Scientific Research, and the Southeastern Center for Electrical Engineering Education for providing me with the opportunity to spend an extremely worthwhile and enjoyable summer at the USAF School of Aerospace Medicine, Brooks AFB, Texas.

I also would like to thank the personnel in Flight Medicine who not only provided excellent working conditions and staff support but were great people with whom to work. Finally, I especially want to thank

Gil Tolan - for his continuing collaboration, guidance, and support, beginning with the refinement of the original idea and throughout the summer;

Bonnie Fridley - for her invaluable assistance in the extensive literature searches required by the project;

George Troxler - for many enjoyable discussions and his helpful suggestions;

Bernice Walters, Margie Lee, and Jackie Chaplin - for typing the several drafts of the technical report; and

Harry Hughes - for making everything run so smoothly for SCEEE Fellows at USAFSAM.

CONTENTS

	Page
INTRODUCTION	5
CORONARY-PRONE BEHAVIOR	5
STUDIES OF AUTONOMIC NERVOUS SYSTEM ACTIVITY	7
Autonomic Balance	7
Stereotypy Versus Stimulus-Response Specificity	10
Relationships Among Physiological, Behavioral, and Personality Variables	12
VARIABLES AND INSTRUMENTATION USED TO MEASURE ANS ACTIVITY	13
Cardiovascular Variables	13
Heart Rate and Heart Period	13
Heart Rate Variability	14
Blood Pressure	14
Pulse Wave Velocity and Transit Time	15
Blood Flow Changes	15
Respiration	17
Electrodermal Variables	17
Electromyography	18
Salivation	19
TASK/CONDITIONS USED TO INDUCE ANS AROUSAL	20
REVIEW AND RECOMMENDATIONS	21
REFERENCES	22

List of Tables

Table		
1	Psychophysiological variables used in cited research studies . .	37
2	Laboratory tasks/situations used in each research study cited . .	42

MEASUREMENT OF CORONARY-PRONE BEHAVIOR AND AUTONOMIC REACTIVITY

INTRODUCTION

The major cause of death in the United States for some years has been coronary disease. A 1979 U.S. Air Force report estimated that one-third of the cardiovascular incidents to active duty personnel result in death or disability, that each death or disability of a flyer costs approximately $495,000, and that the total direct costs of cardiovascular disease to the Air Force are approximately $64 million per year (204).

Traditionally, a number of risk factors have been associated with cardiovascular disease (e.g., age, smoking, cholesterol level). Since the early 1960s, however, increasing evidence suggests that the way an individual typically responds to potentially stressful situations is related to the development of atherosclerosis and myocardial infarction. This report reviews the literature on the Type A coronary-prone behavior pattern and suggests directions research should take to further refine the assessment of coronary-prone behavior.

Obtaining reliable and valid measures of coronary-prone behavior suitable for large-scale screening, and not reliant on self-reporting techniques, has special significance for the Air Force. Pilots represent a highly educated, physically fit population that is highly motivated not to disclose or display any symptomatology that might result in nonflying status. Each year the average age of pilots tends to increase (at present, 44% of USAF pilots are 35 or older), and flying becomes more demanding as planes become faster and more sophisticated. New assessment procedures therefore become increasingly important to ensure that pilots are physically and emotionally able to handle these increasing demands.

CORONARY-PRONE BEHAVIOR

The concepts of coronary-prone behavior and stress responses as major factors in the development of coronary disease have received wide support. Typically, this behavior is characterized as--

(1) an overdeveloped sense of time urgency
(2) excessive desire to control the environment
(3) a highly developed need for competitive achievement-striving
(4) aggression or hostility.

Individuals with such behavior perceive their environment primarily in terms of challenges and frustrations, and hyperrespond to these stressors. The precise psychophysiological mechanisms underlying these relationships are at present poorly understood; however, a major component appears to be situational hyperreactivity or hyperlability of the autonomic nervous system (1-5).

Friedman and Rosenman (6,7) have named the profile described above the "Type A Coronary-Prone Behavior Pattern." It is typically measured by the Structured Interview (8) or the Jenkins Activity Survey (9,10). Research employing these measures has demonstrated Type A behavior relationship with myocardial infarction in numerous prospective and retrospective studies (11-14) and with degree of coronary artery occlusion as assessed by coronary angiography (15-19).

Type A individuals have been shown to differ from Type B's on several biochemical measures, including higher serum cholesterol (20,21), higher triglycerides (22), faster clotting time (20), and increased urinary and serum norepinephrine (23,24). Type A's have also been reported to have higher situationally induced changes in blood pressure (25-30) and heart rate (26,27) and to differ from Type B's on a variety of behavioral variables including fatigue suppression (31), preference to wait in a group and work alone (27), working at faster rates in absence of deadline (32), and performing relatively poorly on tasks requiring slow response rates (33).

Not all researchers, however, have obtained the expected A/B differences (34); and a few investigators have attempted, with mixed success, to subdivide the Type A category by using additional variables. For example, Scherwitz (35) found that A's who habitually referred to themselves frequently exhibited higher task-related increases in blood pressure than A's who did not. Manuck (27) studied task-related blood pressure and heart rate changes of internal and external locus of control Type A's and Type B's, but found few differences. The vast majority of current research, however, operationally defines coronary-prone behavior solely in terms of the Structured Interview (SI) and/or the Jenkins Activity Survey (JAS).

Although the use of the SI and the JAS has focused attention on the importance of behavior and stress reactions in the development of cardiopathology, defining coronary-prone behavior patterns solely in terms of these two measures has led to methodological problems. Specifically, the Structured Interview produces a five-category sort (A1, A2, X, B3, B4), with approximately 60-70% of most populations being A1 or A2. In addition, the divisions between the categories are highly subjective and NOT operationally defined, leading to relatively poor interrater and intrarater reliability, especially for a five-category sort. The JAS avoids these two problems but creates two others. The current version (Form C, Psychology Corp., 1980) is a pencil and paper test consisting of 52 multiple-choice items. Answers are differentially weighted on the basis of factor loadings, and the test generates four scores--each based on a subset of 13 different questions. Scoring, therefore, is completely objective, but validity of the scores requires subject self-awareness and honesty. At best, these assumptions are dubious when applied to most populations; at worst, the test scores are easily manipulated by a person of average intelligence with a casual understanding of Type A behavior.

Since autonomic reactivity is assumed to be a major mechanism linking coronary-prone behavior and cardiovascular pathology, refining the measurement of coronary-prone behavior by systematically investigating individual differences in autonomic responsivity seems more logical than refining the measurement of voice stylistics and mannerisms (the basis of the SI assessment) or attempting to increase self-awareness and honesty in self-reporting (implicit in the JAS).

Research on risk factors and coronary disease has been largely validated in terms of atherosclerosis and coronary angiography. Many individuals, however, die suddenly with minimal coronary artery occlusion--suggesting vasospasm and/or electrochemical malfunction rather than physical obstruction as the major underlying factor. Several recent theoretical and review articles (36-40) have focused on psychological factors and autonomic nervous system responses to stress, and the relationships of these variables to vasospasm and "sudden death," thus supporting the strategy of approaching coronary-prone behavior in terms of individual differences in autonomic responsivity.

STUDIES OF AUTONOMIC NERVOUS SYSTEM ACTIVITY

For convenience, the discussion of studies of autonomic nervous system (ANS) activity will be divided into three categories:

(1) Autonomic balance or dominance between the sympathetic and the parasympathetic branches

(2) Individual-response specificity (stereotypy) versus stimulus-response specificity

(3) Relationships among physiological, behavioral, and personality variables in one or more specific populations.

Autonomic Balance

Contemporary research on balance within the autonomic nervous system derives from Eppinger and Hess' concept of vagotonia and sympathicotonia first published in 1910 (41). They postulated that normal body functioning represented a balance between the mutually antagonistic sympathetic (SNS) and parasympathetic (PNS) branches of the ANS and that imbalance would account for a variety of disorders. This model guided Wenger's classic work on psychophysiological measurement of the ANS during the 1940s and 1950s (42-45). He restated their original hypothesis in 1941, using chemical rather than anatomical differentiation for the branches of the ANS as follows (42):

> The differential chemical reactivity and the physiological antagonism of the adrenergic and cholinergic branches of the autonomic nervous system permit a situation in which the action of one branch may predominate over that of the other. This predominance, or autonomic imbalance, may be phasic or chronic, and may obtain for either the adrenergic or the cholinergic system.
>
> Autonomic imbalance, when measured in an unselected population, will be distributed continuously about a central tendency which shall be defined as autonomic balance.

For a summary of earlier work on the psychophysiology of the ANS, see Darrow's excellent 1943 review article (46).

Wenger's strategy was to measure several noninvasive psychophysiological variables (e.g., dermographia, salivary output, heart period, skin conductance, respiration period, systolic and diastolic blood pressure). Each of these measures was interpreted in terms of a continuum of sympathetic-parasympathetic dominance. He hypothesized that

(1) these measures would be highly intercorrelated (i.e., increased heart rate, blood pressures, perspiration with a high degree of vasoconstriction, and decreased salivation would indicate sympathetic dominance over the opposite responses, parasympathetic, and

(2) individuals would be normally distributed along this continuum: the central portion of the distribution corresponding to persons in "autonomic balance," with individual scores clustered about the mean scores on each variable; and scores clustered either in the lower or upper end of the continuum representing persons in "imbalance."

To Wenger's consternation, almost two-thirds of the 632 young adult males tested in 1944 showed mixed patterns not consistent with this model (44). Some of his later work discusses "mixed" patterns, and five patterns (the three stated at the first of this section plus two mixed patterns--beta and Tb) have been reported (47,48). Wenger's research, however, continued to focus on the idea of a single continuum of "scores of autonomic balance" ($\bar{A}$), and his factor analyses of the data were directed toward refining a regression equation for the assessment of $\bar{A}$--a general factor of ANS activity under resting conditions.

The most recent published version of Wenger's equation (45) computes $\bar{A}$ by taking the raw data from seven tests (persistence of red dermographia, salivary output, heart period, standing palmar conductance, volar forearm skin conductance, respiration period, and pulse pressure), converts them into standard T scores, multiplies them by their beta weights, and then adds these weighted standard scores. For most populations, this procedure produces a distribution of $\bar{A}$ with a mean and standard deviation of approximately 70 and 7; low scores are interpreted as sympathetic dominance, and high scores as parasympathetic dominance. It is worth noting that the sum of the beta weights for heart period and standing palmar conductance are equal to the sum of the beta weights for the other five variables combined. The $\bar{A}$ score, therefore, is heavily weighted by these two variables.

This approach, the determination of a regression equation for a single general factor, is by its unidimensional nature restricted in its ability to distinguish among members of a population suffering from a variety of multifaceted disorders. If the research goal is to distinguish among individuals on the basis of autonomic activity, this strategy appears both restrictive and simplistic when compared with a multifactor model.

During the 1960s Wenger's research interests shifted to include the study of ANS activity during sexual arousal. He noted ". . . the concept of autonomic balance, i.e., relative SNS or PNS dominance, which has been profitably applied to the examination of comparatively stable conditions in groups (Wenger 1966), may not be particularly relevant to complex and highly specialized emotional episodes such as sexual excitement." (49) Nevertheless, his 1972 article, summarizing more than two decades of research, contains the following ". . . the antagonistic control of most ANS functions by the two branches of the system automatically sets the stage for the appearance of a general factor of autonomic function." (45)

Some of the Wenger group's most recent research on autonomic balance in young women makes reluctant and limited use of a multifactor approach (50). In their factor analysis of the data, Lucio and Wenger made presumptive decisions about the underlying physiological functions and then selected one measured variable (which they felt was representative) for each of these underlying functions. Analysis of the 10 selected variables produced six factors which Lucio and Wenger suggest "are not readily interpretable," and one very weak general factor. An interesting finding, however, is that heart period had the highest or second highest loading on three of these six factors.

Cattell (51), on the basis of additional factor analysis of Wenger's data and data from numerous other human and animal studies of psysiological and behavioral variables related to autonomic function or emotionality, suggests a three-factor model: a general autonomic activity factor identical with anxiety and two other autonomic activity factors--sympathetic activity and parasympathetic activity--which he notes appear to be more states than traits. This latter suggestion by Cattell is consistent with recent work on Type A coronary-prone behavior, which has demonstrated that the greatest psychophysiological differences between Types A and B are situationally induced reactive phenomena (26-28). If a state model of ANS activation is valid, then Wenger's approach, which is primarily based on data obtained under passive relaxed conditions, would fail to distinguish among individuals on the basis of their ANS functioning. Although most of Wenger's studies did not include muscle tension/motor activity measures, Cattell's analyses of related studies suggest a fourth factor, a "motor discharge" factor.

More recently a general-factor approach to ANS functioning has been adopted by Porges (52), who has suggested a technique to assess autonomic balance based on the covariance of heart rhythm and breathing. In essence, Porges' approach conceptualizes heart rhythm as an additive model of three components--sympathetic influences, parasympathetic influences, and effects intrinsic to the heart, the latter being assumed to be random relative to the fluctuations of the SNS and PNS. Respiratory changes are assumed to be completely vagally innervated and are used to estimate the PNS contribution to heart rhythm by using a weighted measure of coherence derived from a cross-spectral analysis of simultaneously recorded heart rate and respiration data. Porges' research applications have been primarily with hyperactive and autistic children. Harrell (53) examined the weighted coherence scores of normal adults in response to a series of signaled 105-dB bursts of white noise. He found that above-the-median-coherence subjects (interpreted as relatively more parasympathetic dominant) tended to have greater stimulus-induced increases in heart rate and less stimulus-induced heart rate variability. These results are not consistent with predictions made from Porges' equation.

A different single-factor approach has been suggested by Patton (54). He created an "Index of SNS" by calculating T scores for each subject on each of four autonomic measures (pulse, systolic blood pressure, instep temperature, and square-root palmar conductance) under relaxed and four task conditions. Using mean scores and lability scores (55) for each task condition (based on a "nonstress comedy" condition as a baseline, he found that relative tc other subjects, individuals responded consistently across task conditions in terms both of changes and of absolute level of sympathetic activity.

Although Wenger's research is the most comprehensive and well-controlled investigation of ANS responses in the literature (and these research efforts span more than a decade of active work), his findings have stimulated surprisingly little additional research. A search of Science Citation Index for the period 1974-80 located 57 articles citing one or more of Wenger's reports, but only one of these articles used the concept of autonomic balance in the sense that Wenger used the construct (56); most were citations to illustrate a minor point in the citing article. This lack of continuing research, however, may in large part be due to the fact that Wenger's work is based on a single general-factor model for ANS functioning and that his data were collected under relaxed conditions.

A somewhat different conceptualization of autonomic balance has been proposed by Gellhorn (57-60), who suggests a two-factor model. In this model activation of the sympathetic branch (ergotropic activity) is associated with action-oriented coping responses, whereas trophotropic (parasympathetic) activity is associated with passive responses. In normals, these two systems are balanced in their ability to respond to any given stimulus. The ANS may be "tuned," however, either ergotrophically or trophotropically; in either case the reciprocal system is less effective. Gellhorn's model predicts that an organism is more apt to respond in accordance with the state of the tuning, even when the stimulus would produce the reciprocal response in a normally balanced organism (i.e., when compared to a balanced organism, an organism that was ergotrophically (sympathetically) tuned would tend to respond with enhanced sympathetic response and diminished parasympathetic rebound to ANY stimulus situation). For example, this model would predict that an individual who had a high-baseline function (e.g., heart rate, 17-OHCS level) and was ergotrophically tuned, would under stress tend to exhibit relatively large increases from the already high baseline; whereas an individual who had a low baseline and was trophically tuned, would tend to decrease from the already low-baseline level. These changes would move the individual further from the population mean.

The Law of Initial Values (61) would predict that population extremes measured under baseline conditions (initial values) would tend to regress toward the mean under stress. (The Law of Initial Values is discussed further in the next section.) In addition to several studies discussed by Gellhorn in the previously cited articles, research by Mason et al. (62,63) on 17-OHCS levels found that some chronically low 17-OHCS excretors tended to suppress their 17-OHCS production even more on stressful days than on average days.

Stereotypy Versus Stimulus-Response Specificity

Some studies of ANS reactivity have attempted to demonstrate individual-response specificity (stereotypy) as contrasted to stimulus-response specificity. The most widely cited proponents of this position are John and Beatrice Lacey (64-69). During the 1950s they conducted an extended series of studies using measures of palmar conductance (resistance), heart rate, heart rate variability, and systolic and diastolic blood pressure, taken during several cognitive-task and noxious-stimulation situations (e.g., cold pressor, hyperventilation, mental arithmetic, word fluency). They concluded that

> For any set of autonomic functions, all Ss exhibit, in response to effective stimulus-conditions, idiosyncratic patterns of autonomic activity. These patterns of response tend to be reproduced from one stressor-episode to another, from one occasion of measurement to another. (69)

One interesting methodological suggestion by the Laceys is that an alerting period (i.e., a quiet announcement of what the next task will be and that the task will begin in 60 seconds) tended to reduce the common factor of arousal generated by a task following a relaxed physiological state. If the Laceys' conclusion is correct, this common anticipatory alerting response may be an example of Cattell's general anxiety factor.

Other studies have focused on similarities of physiological responses across subjects, usually to specific emotion-provoking stimuli or sensory conditions (stimulus-response specificity, or "specificity"). The most widely researched stimuli are associated with subjective reports of fear and anger, and several researchers have demonstrated differential physiological response patterns to these two stimuli (70-72). Additional situations include those producing sadness or mirth (73), sexual arousal (49,74), and prolonged stay at altitude (75). I believe, however, that to seek either stereotypy or specificity creates a false dichotomy, not unlike the futile attempts several years ago to attribute given behavior to either heredity or environment. Both logic and available data suggest that although individuals have relatively consistent idiosyncratic response patterns, they also show similarities to a given set of conditions. Therefore, depending on the homogeneity of the populations and the quality and intensity of the stimulus situations that are compared, it is possible in a given study to emphasize either individual differences or similarities. For example, although Sersen (76) found stereotypy in his study of skin conductance, heart period, respiration, and blood pressure across six stimulus conditions, he also found stimulus specificity--which does not seem surprising, considering the conditions he used. Essentially, he found that subjects' response patterns to a tape recording of a poem, a tape of classical music, and a white field illuminated by a 60-W light bulb were similar and that they differed from responses (also similar to one another) to 100-dB sound, a cold pressor test, and difficult math calculations.

In addition, however, Sersen demonstrated that data analysis (i.e., task scores versus change scores, and whether a regression model or a covariant model is used to analyze changes from the baseline) can markedly alter the relative contributions attributed to stereotypy or specificity (77,78). The best method of analyzing change scores, however, is still open to debate. The argument in favor of a covariant analysis of changes from the baseline (initial value) derives largely from the Law of Initial Values (LIV)--which states that the higher the initial level of a function, the smaller the response to function raising and the greater the response to function lowering (61). More recently, however, Gellhorn's two-factor model (discussed in the previous section) suggests that under stress, individuals with high initial values may show exaggerated increases and individuals with low initial values may show exaggerated decreases. If the LIV holds only for the central portion of a population distribution and Gellhorn's model holds for many of the individuals in the tails of the distribution, then data analysis based on task means or simple change scores (task means - baseline means) analyzed using simple comparisons or ANOVA, may be more useful than covariant analysis.

Relationships Among Physiological, Behavioral, and Personality Variables

Numerous studies have been conducted comparing two or more populations on numerous physiological, behavioral, and personality variables, but most are of limited usefulness since the populations being compared are themselves extremely heterogeneous (e.g., psychiatric patients versus normals, neurotics versus psychotics). Also, many personality variables and behavioral items, themselves interrelated, are typically correlated with a variety of physiological measures, leading to spurious relationships that are difficult to separate from actual relationships. Here, the most extensive and systematic data are those of Wenger (43). In addition to $\bar{A}$ scores, he reported data on individual variables for several subject populations, including hospitalized psychoneurotic patients (by diagnostic category), ulcer patients, and "operational fatigue" patients.

Following are some typical results of studies comparing two or more patient populations. Sterman et al. (79) found that patients with very prolonged EEG responses to a cold pressor test, compared with patients having very brief EEG responses (recovery toward baseline began during stimulation), tended to show prolonged autonomic responses and be characterized by higher heart rate levels and relatively lower levels of skin conductance.

Shipman et al. (80) used a somewhat different approach. They categorized outpatients on the basis of the response organ system showing the highest consistent relative response (i.e., muscle, heart rate, blood pressure, GSR) and reported differences among these four groups on a variety of personality measures; e.g., muscle responders were older and tended to be more depressed.

The most common approach, however, has been to select a number of variables and analyze them in terms of a correlation matrix. The following two studies illustrate this approach. Zuckerman et al. (81) examined the relationships among anxiety, depression, hostility, GSR, heart rate, and breathing during a cold pressor test for normals and psychiatric patients and noted a variety of relationships that were difficult to interpret (e.g., patients had higher breathing and pulse rates, normals had higher systolic blood pressures). Similarly, Kelly et al. (82) examined anxious patients and normal controls using forearm blood flow, EMG, heart rate, skin resistance, and a battery of personality measures and found that patients had higher forearm blood flow and heart rates and that forearm blood flow was correlated with manifest anxiety. Most relevant for the present review, however, are the differences between Types A and B, which were discussed in the first section.

In general, relatively less attention has been given to the simultaneous recording of several psychophysiological measures of ANS activity during the 1970s than during the 1950s or 1960s. At first this seems surprising since the earlier research provides an excellent foundation for further investigation of noninvasive measure of ANS activity, utilizing the significant improvements in instrumentation and computer interfacing and availability that have occurred during the last decade. I believe that this lack of basic research largely results from an increased emphasis on the clinical applications of biofeedback. Within the last decade, clinical biofeedback has become extremely popular. It is rapidly becoming generally accepted in the treatment of numerous disorders ranging from Raynaud's syndrome, where it is considered by many to be the most consistently successful therapeutic intervention (83-85), to migraines (86,87) and muscle rehabilitation where it has become an invaluable diagnostic and therapeutic technique (88,89).

In addition, as our understanding of the complexity of the ANS has increased, several researchers have become highly specialized, focusing on one or two psychophysiological variables or on a particular task or set of conditions. For example, Burdick (90-94) has conducted numerous studies on heart rate; Obrist et al. (95-100) have worked primarily with measures of blood pressure and blood flow; and Lovallo et al. (101-103) have studied responses to the cold pressor test.

The next two sections examine the most widely used measures of ANS activity individually and the tasks and situations used to induce physiological arousal.

VARIABLES AND INSTRUMENTATION USED TO MEASURE ANS ACTIVITY

Five categories of psychophysiological variables associated with sympathetic and parasympathetic arousal will be discussed:

(1) Cardiovascular Variables
(2) Respiration
(3) Electrodermal Variables
(4) Muscle Tension
(5) Salivation

The intent of this report is to review research on noninvasive measures of coronary-prone behavior and autonomic activity, so measures requiring the drawing of blood samples are not included. Although endocrine measures of ANS activity may be obtained from urine specimens (e.g., epinephrine, norepinephrine, metaepinephrines, VMA, and 17-OHCS), it seemed more logical to discuss both serum and urinary measures of psychoendocrine activity in a separate report. Psychoendocrine measures, therefore, are not considered in this report.

Cardiovascular Variables

This category includes not only the largest number of variables used in psychophysiological research but also the most widely used ones. The most common cardiovascular measures are heart rate, blood pressure, transit-time, and blood flow change charted by photoplethysmography and skin temperature.

<u>Heart Rate and Heart Period</u>--Heart rate (HR) is traditionally defined in terms of the number of ventricular contractions, or beats, per minute (bpm). When the measurement is taken for 30 seconds or longer, the number of beats are counted and used to compute bpm in straightforward fashion. If on the other hand, the heart period (HP), the time interval between two successive beats, is used to compute the HR in bpm, the term "HR" can be misleading since it implies a tonic measure when in fact a highly variable phasic measure is being observed and recorded. Some instruments that monitor the interbeat interval (HP) and display HR in bpm offer the researcher options (e.g., the HR being calculated on the basis of either two successive beats or five beats--the average of four successive HPs) that further complicate the situation.

Heart Rate Variability--Unlike HR and HP, which have fairly standard definitions, heart rate variability (HRV) is computed and reported in several different ways, creating considerable confusion when data is compared across different methods. Two basic issues are involved in HRV:

(1) the time interval underlying the HR measurement itself, and
(2) the statistical procedures used to calculate HRV.

With regard to time intervals underlying the HR measures, at one end of the continuum is the HP data and at the other end is HR counted over 60-second or longer intervals. Two researchers will generate quite different values of HRV. For example, during a 20-minute task one researcher will measure HP periodically throughout the task, use these HPs to calculate HR (HR = 60 sec/HP), and then calculate the variance (HRV); while a second researcher will periodically count the number of beats in 60-second intervals and use these HRs to calculate the variance (HRV).

In addition to computing HRV as the variance (or standard deviation) of the individual measures, six other methods of determining HRV are discussed by Burdick (93). The most popular of these are the mean-square successive difference (104,105), the auto-correlation-lag-one (the product moment correlation of each observation with its successor), and the coefficient of temporal variability (the ratio of the square root of the mean-square successive difference divided by the mean) which is favored by Burdick (90-94).

Blood Pressure--Measures of blood pressure are the second most common cardiovascular variables reported in the literature. Clinical diagnosis always makes use of both the systolic and diastolic pressures, but because of their covariance, research has often used one or the other or the "mean" blood pressure. In the past, diastolic was more widely used; however, systolic is now more popular.

One major disadvantage of noninvasive sphygmomanometric techniques for monitoring blood pressure is that they do not provide continuous readings, as an arterial catheter can. Several researchers have developed monitoring devices with an automatic inflating/deflating cuff containing a built-in microphone to detect the Korotkoff sounds (106-108). One system, designed by Obrist (109), employs two cuffs (one on each arm) that automatically inflate/deflate in sequence, with one cuff measuring systolic and the other diastolic. These devices are useful, especially the portable ones that can be worn by the subject throughout a normal day or night; however, they permit only periodic measurement of blood pressure and in general are more prone to error (especially of diastolic readings) than auscultation by a trained technician using a regular sphygmomanometer.

In an attempt to provide continuous readings using sphygmomanometric techniques, Tursky et al. (110) developed a system that inflates the cuff to a predetermined value near either the normal systolic or diastolic pressure. The presence or absence of the Korotkoff sounds detected by a cuff microphone indicates whether the blood pressure is above or below the preset cuff-inflation value. The price paid for these continuous readings, however, is substantial, because the information obtained is binary (above or below the preset value) and, since the cuff remains inflated during the entire period when readings are taken, readings can only be obtained for short periods of time before the cuff must be deflated to allow blood circulation into the arm. During the 1970s, in an attempt to provide a continuous measure of blood pressure changes for biofeedback training and psychophysiological research, several researchers began working with arterial pulse wave velocity.

Pulse Wave Velocity and Transit Time--The pulse wave velocity (PWV) is the rate the pulse pressure waves travel through the arteries and is measured by recording the times at which pressure waves reach two points on one major artery (e.g., the brachial and radial pulses), subtracting the times, and dividing by the distance between the two. In actual noninvasive measurement, however, it is difficult to obtain continuous trains of artifact-free pulses from two different sites. Measurement is also complicated by the fact that the time differential is very short. Most researchers, therefore, prefer to eliminate one of the pulse detectors and use the time differential between the R wave of the ECG and a single distal location where a strong pulse can be obtained (typically radial, carotid, or temporal). This time interval is referred to as pulse transit time (PTT) or simply transit time (TT). Although TT covaries with PWV, the relationship is not perfectly linear since TT is a function not only of the PWV but also of several intracardiac events (e.g., electrical depolarization, opening of the semilunar valves, and expulsion of the blood into the aorta) (111-114).

In 1976, Steptoe et al. (114) reported linear correlations of -.91 to -.98 between mean blood pressure (obtained from an arterial catheter) and TT. Although his work generated considerable interest, more recent work has found substantially lower correlation coefficients, with large individual differences in the magnitude of the correlations between TT and both systolic and diastolic blood pressure (96,115).

The fact that transit time is not highly correlated with other commonly used cardiovascular measures may turn out to be advantageous, since TT provides another noninvasive measure of cardiovascular functioning.

Blood Flow Changes--Noninvasive measurement of changes in blood flow (often referred to as pulse volume changes; e.g., finger pulse volume) pose major methodological problems. Traditionally blood flow has been measured using volume plethysmography, skin temperature, and more recently, ultrasound. Currently, three major types of plethysmography (volume change measurement) are used to measure blood flow changes:

(1) Volume plethysmography, which measures changes in the volume or girth of a limb

(2) Impedance plethysmography, which measures resistance to an electric current

(3) Photoplethysmography, which measures the amount of transmitted or back-scattered light from a constant light source.

Volume plethysmography consists of encasing a portion of the limb or digit in a rigid, fluid-filled container that is connected to a device to measure/record fluid displacement. Simple in design, this procedure has been used since the late 1800s (116). Although simple in theory, in practice it poses three major methodological problems:

(1) Movement artifact is very difficult to control

(2) The output reflects the sum of blood flow changes in both skin and muscle, which may be in opposite directions

(3) Unless very carefully controlled, the transducer produces temperature-induced changes in blood flow in the limb (117).

These problems can be somewhat overcome by using the tip of a finger (102,118). Volume plethysmography is occasionally employed to measure forearm blood, often using the venous occlusion technique (81,119).

One unusual application of volume plethysmography is the measurement of vaginal engorgement as a function of sexual arousal; however, even here recent work seems to be shifting toward photoplethysmography (74,120).

Girth plethysmography using strain gauge transducers (flexible tubes--filled with electrically conductive liquid, gel, or paste--wrapped snugly around the appendage) or for some applications a bimetallic C-clip, avoids the temperature control problem of traditional volume techniques. The transducers are simpler to design and control, but they are also very sensitive to movement artifact (121). Girth plethysmography is widely used to measure respiration; another popular contemporary application is measuring penile tumescence either as a function of sexual arousal or during REM sleep (122,123).

Impedance plethysmography consists of passing high-frequency alternating current (AC) through tissue and measuring the impedance. Impedance, the living-tissue opposition to AC, includes not only resistance properties (measured when direct current is used) but also capacitance properties (124). Brown's review of psychophysiological instrumentation suggests that compared to the volume/girth techniques, impedance plethysmography "permits essentially the same measurement . . . is relatively insensitive to motion artifact, requires much simpler instrumentation, and permits greater subject comfort" (117). In spite of these accolades, photoplethysmography has been more widely used during the 1970s.

Photoplethysmography directs a small constant light (usually in the red and infrared wave lengths) into tissue and measures either the amount of backscattered light or, if the light is able to pass through the tissue (e.g., earlobe, fingertip), transmitted light (the transillumination technique) with a photocell detector. Blood volume changes produce changes in the amount of light received by the photocells. Photoplethysmography has the advantage of requiring only a small transducer, and by using fiberoptic light guides, the backscattering technique can be used in almost any body location (117,124). Backscattered or transilluminated techniques have been used in several recent psychophysiological studies (125-127).

Skin temperature is undoubtedly the simplest and most widely used current technique for measuring surface blood flow changes. It is popular not only for research but also for a number of clinical applications (83,86,128). The most commonly used transducers are small plastic-coated spherical thermistors which can be easily taped to any body surface. (The same thermistors can also be used in any body orifice.) Unlike previously discussed plethysmographic techniques, skin temperature does not provide a measure of beat-to-beat pulse volume changes; instead, it reflects longer term gradients of blood flow changes taking place over periods of several seconds to several minutes.

Doppler flowmeters are the newest of the techniques discussed. They have rapidly become popular for clinical diagnosis because of their availability and the ease with which they can be used (129). These instruments emit an ultrasound beam of 2-10 MHz which is transmitted into the tissues via an acoustic gel on the skin. A small portion of the sound is reflected back by the moving red cells, and the frequency of the returned signal increases as a function of the velocity of the red cells, the Doppler shift (129,139). Although widely used clinically, very few psychophysiological research studies have used Doppler flowmeters.

Respiration

Ideally, measurement of respiration should include time of inspiration and expiration and the amount of air exchanged. Since the volumes of air involved in each cycle and the flow rates are small, it is difficult to accurately monitor the airflow without obstructing an individual's natural breathing pattern. Psychophysiological research, therefore, has tended to neglect respiratory measures, and when they are included, to generally measure just respiratory rate, using a chest strain-gauge transducer similar to the ones discussed in the section on blood flow (117).

Wenger's study (43) of young Air Force males where he obtained spirograms on each subject and analyzed them for several respiration variables is virtually the only study that includes several respiration variables. In both his earlier and later studies, Wenger (45) used just a strain gauge to obtain respiration period.

Electrodermal Variables

Until as recently as the early 1960s, measures of skin potential (SP--the electrical potential difference between an active and a reference skin site) and skin resistance (SR--the resistance to current flow when a weak direct current is passed between two electrodes) were considered manifestations of the same phenomenon. SP and SR are now generally accepted as different, albeit related, phenomena, although their exact nature is still under discussion.

Instrumentation for measuring SR or skin conductance (SC--the reciprocal of SR) uses one of two circuit arrangements:

(1) constant voltage, which measures changes in skin resistance as fluctuations in current, or

(2) constant current, which measures skin resistance changes as voltage fluctuations (117).

Although not as commonly used, if AC is substituted for DC, the resulting electrical resistance is called skin impedance (SZ) since it includes both resistive and capacitive elements.

Skin resistance measures (SR, SC, SZ) may be measured from any two skin sites. Although palmar and plantar surfaces are among the most active sites for skin resistance measures and SP, several other sites also exhibit high activity (e.g., forehead and medial portion of the foot) (131); under stress, generally inactive areas (e.g., chest and arm) may exhibit high activity (132).

Skin potential would ideally be measured with one electrode on the skin surface (at an active location) and the second electrode inside the body. Since this is not generally feasible, it is common practice to select a second, inactive skin site (e.g., earlobe or inner arm near elbow) for the reference. Activity at these sites can be further reduced by breaking through the outer skin layer and making contact with subdermal layers, using procedures such as skin abrasion with a gritty salt paste or tiny punctures with a fine-grade needle (133). Note, however, that if an inactive site (as opposed to inside the body) is selected, there is always an absolute error with reference to inside the body. Thus, the standardization of inactive-site electrode placements is greatly to be desired.

Electrodermal measures (still often referred to by the generic term "galvanic skin responses," GSR) have traditionally been widely used. With the popularity of clinical biofeedback during the 1970s, however, they have been somewhat overshadowed by electromyography (EMG) and skin temperature. For a summary of electrodermal research and a discussion of the electrical properties of the skin, see Edelberg's excellent 1972 review (133).

Electromyography

As with skin temperature, EMG is essentially a fairly straightforward variable which has become widely used. EMG is essentially a measure of the electrical activity resulting from the progressive change in polarization of the surface membrane of the muscle fiber, and is typically measured noninvasively by multiple relatively large (e.g., 4-mm to 4-cm-diameter) surface electrodes.

Muscle action potentials can be recorded from any muscle group in the body. Several researchers have examined large numbers of muscle groups in an attempt to determine whether there are general muscle factors and which muscles best reflect these factors. Although there is considerable intraindividual variation, under resting conditions, residual muscle tension tends to be found primarily in the limbs, with little electrical activity in the head and neck muscles; under mental workload, neck and muscle tension typically increases significantly. This is undoubtedly one reason that the frontalis (more precisely, the center of the forehead) has become the most popular site for electrode placement in studies measuring general arousal. Also, the forehead is easily accessible and socially acceptable; and although a subject can easily wrinkle his forehead and see the increase in muscle activity, the electrode measures the subtle, largely involuntary muscle activity associated with facial expression.

The frontalis placement, however, is by no means universally accepted as the best site for measuring total body muscle tension. For example, some studies suggest that with individuals under stress and frustration, the most prominent muscle tension was found in the trapezius and masseter muscles.

Unlike the slow temperature gradients, the electromyogram characteristically consists of bursts of spikey activity followed by (or alternating with) periods of relative quiescence. Consideration of both the mean value and the variance for the observation period is therefore desirable (134).

Salivation

Research on salivation, as compared to that on the other ANS variables, has been largely ignored in America and has fared only slightly better in England and Australia. Perhaps research on salivation has not been popular because it is not seen as having much clinical utility; however, this is unfortunate because relatively high test-retest reliabilities are typically reported and studies have suggested relationships between salivation and arousal (135,136), introversion (137,138), and affective disorders (139,140).

The only researcher who appears to have studied salivation as part of an ANS profile is Wenger (42,43). See Brown (141) for an extensive and very readable review of the literature, and White (142) for an update.

White also compared measurements of salivation, using three noninvasive techniques:

(1) Wenger's procedure, where saliva is collected from the whole mouth by a small tube held between the lips and connected to a vacuum collection jar

(2) A parotid capsule suction technique in which the end of the collection tube is a small capsule that is positioned over the Stetson duct and held in place by an outer suction chamber

(3) Cotton swabs placed sublingually and buccally.

The results obtained from the three methods tended to be highly intercorrelated, suggesting that all three procedures are useful and that the choice among them can be based on convenience, cost, the exactness of the measurement desired, and whether secretion rate changes during the collection period are desired (142).

Table 1 provides a cross-tabulation of research studies cited in this report with each variable. For additional general background in the area, Greenfield's and Sternbach's Handbook of Psychophysiology is highly recommended (143). Although the Handbook is becoming dated (published in 1972) and does not reflect the tremendous development of instrumentation that has taken place during the 1970s or adequately cover recent topics (e.g., transit-time, Doppler flowmeters), it still remains the best single source of information. For recent reviews of noninvasive diagnostic techniques for cardiovascular disease, see Barnes (130) and Lieberman (129).

TASKS/CONDITIONS USED TO INDUCE ANS AROUSAL

Numerous laboratory and field situations have been used to produce ANS arousal (stress). Although this report is primarily concerned with laboratory stressors, the following will illustrate some of the many and varied real-life situations that have been used. For example, studies have been conducted on test pilots before and after flying (144); racing-car drivers during a race (145); patients awaiting surgery (146); UDT teams before a stressful underwater swim (147); commuters, as a function of traffic congestion and length of daily commute (148); and the most popular, students before oral or written examinations (149-151).

Since laboratory situations are directly manipulable by the experimenter, the investigator must determine a number of parameters, including the following:

(1) Should the task/situation involve aversive stimulation?

On the plus side, aversive stimulation typically produces greater physiological change; however, the major deterrent is that the stimulation may not be acceptable to many potential subjects.

(2) Should the task be active or passive?

Recent work by Obrist et al. (97,99) on cardiovascular interactions suggests that during passive tasks the heart is under vagal (parasympathetic) dominance, whereas during active coping tasks the heart is primarily under the beta-adrenergic (sympathetic) control. They conclude that the greatest task-related changes in cardiovascular activity are produced by tasks that engage a subject and provide him some measure of control over the task. Also, recent research on coronary-prone behavior suggests that Type A-Type B differences are likely to be greater under challenging reactive task conditions (25-29, 35,152).

Active coping, however, when combined with aversive stimulation, assumes that the subject has (or perceives to have) some degree of control over the occurrence of the aversive stimulus. This introduces the question of whether control over aversive stimulation increases or reduces physiological arousal, as compared to lack of control over such stimulation. Although these phenomena are complex (see Averill (153) for a summary of the literature on personal control and aversive stimuli), in general, control with a high degree of uncertainty seems apt to produce higher levels of arousal, both subjective and physiological, than either highly predictable personal control over the aversive stimulation or no control (passive aversive stimulation). This assumption is also supported by a recent study in which Type A and Type B women were asked to report fatigue and symptoms associated with ANS arousal during a task involving unpredictable and predictable noise. Greater numbers of symptoms were reported by both A's and B's when the noise was unpredictable (154).

(3) If the task is active, should it

(a) be primarily cognitive or psychomotor?
(b) be easy, difficult, or impossible?

(c) involve competition with another person, a computer, a set of standards (actual or deceptive), the subject's own performance, or against the clock?

Virtually all combinations of the above options can be found in the literature (usually with several permutations). Table 2 lists a number of these studies as a function of the task conditions used to induce stress. There is also an overlapping, but separate, literature dealing with "mental workload." For a review of this literature see Moray's book, Mental Workload, Its Theory and Measurement (155), or Wierwille's review article (156).

REVIEW AND RECOMMENDATIONS

Strong, continually increasing evidence supports the concepts of coronary-prone behavior and stress as major factors in myocardial infarctions and the development of atherosclerosis. The most probable underlying physiological mechanisms involve hyperreactivity and/or hyperlability of the sympathetic branch of the autonomic nervous system. This hyperreactivity and/or hyperlability appears to be a reactive (state) phenomenon.

Early research on Type A and Type B differences demonstrated that Type A's tend to manifest higher reactive levels on several serological variables (e.g., cholesterol, norepinephrine) associated with accelerated atherosclerosis. More recent research has also shown Type A's to be more reactive on several psychophysiological variables (e.g., heart rate, systolic blood pressure) under a variety of task conditions.

These findings are more impressive than a review of the studies would suggest when we consider the methodological problems inherent in using currently available instruments (Structured Interview, Jenkins Activity Survey) to assess coronary-prone behavior. Problems such as low interscores reliability, lack of subject self-awareness, and attempts to deceive by the subject, result in considerable potential misclassification. These problems become even more important if the individuals being classified are of above-average intelligence and highly motivated not to display coronary-prone behavior. Air Force pilots would clearly fit this description. For the value of the coronary-prone behavior pattern as a predictor of coronary disease to be fully realized, new ways of assessing the pattern must be developed to avoid these problems.

To date there has been little systematic attempt to examine multiple autonomic variables associated with the coronary-prone behavior pattern. What research has been done has tended to focus more on the task and situation parameters than on profiles or autonomic responses.

I therefore recommend that, as a logical extension of the current work on coronary-prone behavior, a systematic examination of autonomic profiles be conducted under challenging or stressful conditions, as a function of Type A and Type B behavior, with the goal of developing new and better techniques for assessing coronary-prone behavior.

Three research strategies are suggested, all based on a test protocol involving a number of measures of ANS reactivity:

(1) Stepwise discriminant analysis of Type A and Type B behavior

(2) An R technique factor analysis in which pairs of measures would be correlated for all subjects, and factor analyzed. The literature would predict a three- or four-factor model of ANS reactivity.

(3) A standard-score profile approach in which a profile of standard scores across the ANS measures would be constructed for each individual. If the number of individuals is large enough, this data could also be factor analyzed using the obverse (Q technique) approach in which pairs of individuals are correlated and the matrix factor analyzed.

The following ANS measures should be included: heart rate, heart rate variability, blood pressure, transit time, one or more measures of blood flow (photoplethysmography and skin temperature are recommended), respiration rate, skin resistance and/or skin potential, muscle tension, and salivation.

One or more of the task situations should be challenging, require active involvement of the subject, and contain an aversive stimulus.

The recommended project would be a valuable first step, but only that; heart disease is the logical end point, not Type A-Type B classification. Therefore, assuming that the protocols of ANS reactivity discussed above can be developed, the second phase should be to use these protocols to examine the ANS profiles of individuals undergoing coronary catheterization and to correlate the profiles with objective measures of coronary artery occlusion.

Potentially such a program will develop a noninvasive screening technique that will not only add greater predictability to existing coronary-risk factor equations, but will also suggest intervention strategies that can be used to modify autonomic responses in stressful situations and thus reduce the negative consequences of those situations.

REFERENCES

1. Herd, J.A. Physiological correlates of coronary-prone behavior. In T.M. Dembroski et al. (eds.). Coronary-Prone Behavior. New York: Springer-Verlag, 1978, 129-136.

2. Williams, R.B., Jr. Psychophysiological processes, the coronary-prone behavior pattern, and coronary heart disease. In T.M. Dembroski et al. (eds.). Coronary-Prone Behavior. New York: Springer-Verlag, 1978, 141-146.

3. Glass, D.C. Stress, behavior patterns, and coronary disease. Am Sci, 1977, 65(2), 177-187.

4. Raab, W. Correlated cardiovascular adrenergic and adrenocortical response to sensory and mental annoyances in man: A potential accessory cardiac risk factor. Psychosom Med, 1968, 30, 809.

5. Hartman, L.M. Behavioral prevention of ischemic heart disease. Can Med Assoc J, 1978, 119(6), 599-604.

6. Friedman, M., and R.H. Rosenman. Association of specific overt behavior pattern with blood and cardiovascular findings. JAMA, 1959, 169, 1289-1296.

7. Rosenman, R.H. Coronary-prone behavior: Introduction. In T.M. Dembroski et al. (eds.). Coronary-Prone Behavior. New York: Springer-Verlag, 1978, xii-xvii.

8. Rosenman, R.H. The interview method of assessment of the coronary-prone behavior pattern. In T.M. Dembroski et al. (eds.). Coronary-Prone Behavior. New York: Springer-Verlag, 1978, 54-59.

9. Jenkins Activity Survey (Form C). Psychology Corporation, New York, 1980.

10. Jenkins, C.D. A comparative review of the interview and questionnaire methods in the assessment of the coronary-prone behavior pattern. In T.M. Dembroski et al. (eds.). Coronary-Prone Behavior. New York: Springer-Verlag, 1978, 71-88.

11. Rosenman, R.H., R.J. Brand, C.D. Jenkins, et al. Coronary heart disease in the Western Collaborative Group Study: Final follow-up of 8.5 years. JAMA, 1975, 233, 872-877.

12. Brand, R.J. Coronary-prone behavior as an independent risk factor for coronary heart disease, In T.M. Dembroski et al. (eds.). Coronary-Prone Behavior. New York: Springer-Verlag, 1978, 11-24.

13. Jenkins, C.D., R.H. Rosenman, and S.J. Zyzanski. Prediction of clinical coronary heart disease by a test for the coronary-prone behavior. N Engl J Med, 1974, 290, 1271-1275.

14. Shekelle, R.B., J.A. Schoenberger, and J. Stamler. Correlates of the JAS Type A behavior pattern score. J Chronic Dis, 1976, 29, 381-394.

15. Krantz, D.S., M.I. Sanmarco, R.H. Selvester, et al. Psychological correlates of progression of atherosclerosis in men. Psychosom Med, 1979, 41(6), 467-475.

16. Blumenthal, J.A., R.B. Williams, Jr., Y. Kong, et al. Type A behavior pattern and coronary atherosclerosis. Circulation, 1978, 58(4), 634-639.

17. Frank, K.A., S.S. Heller, D.S. Kornfeld, et al. Type A behavior pattern and coronary angiographic findings. JAMA, 1978, 240(8), 761-763.

18. Jenkins, C.D., S.J. Zyzanski, T.J. Ryan, et al. Type A responses as identifiers of severe atherosclerosis. J Consult Clin Psychol, 1977, 45(6), 1060-1067.

19. Zyzanski, S.J., C.D. Jenkins, T.J. Ryan, et al. Psychological correlates of coronary angiographic findings. Arch Intern Med (USA), 1976, 136(11), 1234-1237.

20. Friedman, M., H.K. Hellerstein, S.E. Jones, et al. Behavior patterns and serum cholesterol in two groups of normal males. Am J Med Sci, 1968, 255, 237-244.

21. Friedman, M., R.H. Rosenman, and S.O. Byers. Serum lipids and conjunctival circulation after fat ingestion in men exhibiting Type A behavior pattern. Circulation, 1964, 29, 874-886.

22. Friedman, M., S. St. George, S.O. Byers, et al. Excretion of cathecholamines, 17-ketosteroids, 17-hydroxycorticoids, and 5-hydroxyindole in men exhibiting a particular behavior pattern (A) associated with high incidence of clinical coronary artery disease. J Clin Invest, 1960, 39, 758-764.

23. Friedman, M., S.O. Byers, J. Diamant, et al. Plasma catecholamine response of coronary prone subjects (Type A) to a specific challenge. Metabolism (USA), 1975, 24(2), 205-210.

24. Dembroski, T.M., and J.M. MacDougall. Stress effects on affiliation preferences among subjects possessing the coronary-prone behavior pattern. J Pers Soc Psychol, 1978, 36, 23-33.

25. Dembroski, T.M., J.M. MacDougall, and R. Lushene. Interpersonal interaction and cardiovascular response in Type A subjects and coronary patients. J Hum Stress, 1979, 5(4), 28-36.

26. Dembroski, T.M., J.M. MacDougall, and J.L. Shields. Physiologic reactions to social challenge in persons evidencing the Type A coronary-prone behavior pattern. J Hum Stress, 1977, 3(3), 2-9.

27. Dembroski, T.M., J.M. MacDougall, J.L. Shields, et al. Components of the Type A coronary-prone behavior pattern and cardiovascular responses to psychomotor performance challenge. J Behav Med, 1978, 1, 159-176.

28. Manuck, S.B., and F.N. Garland. Coronary-prone behavior pattern, task incentive, and cardiovascular response. Psychophysiology, 1979, 16(2), 136-142.

29. Manuck, S.B., S. Craft, and K. Gold. Coronary prone behavior and cardiovascular response. Psychophysiology, 1978, 15, 403-411.

30. Sparacino, J., S. Hansell, and K. Smyth. Type A (coronary-prone) behavior and transient blood pressure change. Nurs Res, 1979, 28(4), 198-204.

31. Carver, C.S., A.E. Coleman, and D.C. Glass. The coronary-prone behavior pattern and the suppression of fatigue on a treadmill test. J Pers Soc Psychol, 1976, 33, 460-466.

32. Burnam, M.A., J.W. Pennebaker, and D.C. Glass. Time consciousness achievement striving, and the Type A coronary-prone behavior pattern. J Abnorm Psychol, 1975, 84, 76-79.

33. Glass, D.C., M.L. Snyder, and J.F. Hollis. Time urgency and the Type A coronary-prone behavior pattern. J Appl Soc Psychol, 1974, 4, 125-140.

34. De Backer, G., M. Kornitzer, F. Kittel, et al. Relation between coronary-prone behavior pattern, excretion of urinary catecholamines, heart rate, and heart rhythm. Prev Med, 1979, 8(1), 14-22.

35. Scherwitz, L., K. Berton, and H. Leventhal. Type A behavior, self-involvement, and cardiovascular response. Psychosom Med, 1978, 40(8), 593-609.

36. Hellstrom, H.R. Coronary artery vasospasm: The likely immediate cause of acute myocardial infarction. Br Heart J, 1979, 41(4), 426-432.

37. Buell, J.C., and R.S. Eliot. The role of emotional stress in the development of heart disease. JAMA, 1979, 242(4), 365-368.

38. Engel, G.L. Psychologic stress, vasodepressor (vasovagal) syncope and sudden death. Ann Intern Med, 1978, 89, 403-412.

39. Boyd, G.W. Stress and disease: the missing link. A vasospastic theory. I. Acute myocardial infarction--a potentially reversible event? Med Hypotheses, 1978, 4(5), 411-419.

40. Dimsdale, J.E. Emotional causes of sudden death. Am J Psychiatry, 1977, 134(12), 1361-1366.

41. Eppinger, H., and L. Hess. Die Vagotonie. Berlin, 1910. (Translation) Vagotonia. New York: The Nervous and Mental Disease Publishing Co., 1917.

42. Wenger, M.A. The measurement of individual differences in autonomic balance. Psychosom Med, 1941, 3, 427-434.

43. Wenger, M.A. Studies of autonomic balance in Army Air Forces personnel. Comp Psychol Monogr, 1948, 19, No. 4.

44. Wenger, M.A., B.T. Engel, and T.L. Clemens. Studies of autonomic response patterns: Rationale and methods. Behav Sci, 1957, 2, 216-221.

45. Wenger, M.A., and T.D. Cullen. Studies of autonomic balance in children and adults. In N.S. Greenfield and R.A. Sternbach (eds.). Handbook of Psychophysiology. New York: Holt Rinehart, 1972.

46. Darrow, C.W. Physiological and clinical tests of autonomic function and autonomic balance. Psychol Rev, 1942, 23(1), 1-35.

47. Wenger, M.A. Studies of autonomic balance: A summary. Psychophysiology, 1966, 2(3), 173-186.

48. Markwell, E.D. An investigation of autonomic balance in tuberculous patients. Psychosom Med, 1961, 23, 392-399.

49. Wenger, M.A., J.R. Averill, and D.D. Smith. Autonomic activity during sexual arousal. Psychophysiology, 1968, 4(4), 468-478.

50. Lucio, W.H., M.A. Wenger, and T.D. Cullen. Psychophysiological correlates of female teacher behavior and emotional stability: A seven-year longitudinal investigation. CSE Ref. No. 44, University of California, Los Angeles, 1967.

51. Cattell, R.B. Handbook of Multivariate Experimental Psychology. Chicago: Rand McNally, 1966, 666-683.

52. Porges, S.W. Peripheral and neurochemical parallels of psychopathology; a psychophysiological model relating autonomic imbalance to hyperactivity, psychopathy and autism. In H.W. Reese (ed.). Advances In Child Developmental Behavior. New York: Academic Press, 1976, 11, 35-65.

53. Harrell, J.P. Individual differences in anticipatory physiological responses to signaled stress: Role of locus of control and autonomic balance. Percept Mot Skills, 1979, 48, 663-669.

54. Patton, G.W.R. Interstressor consistency of subject differences in autonomic activity. J Psychol, 1969, 72, 199-209.

55. Lacey, J.I. The evaluation of autonomic responses: Toward a general solution. Ann NY Acad Sci, 1956, 67, 123-164.

56. Parker, D.M., W.E. Wilsoncroft, and T. Olshansky. Relationship between life change and relative autonomic balance. J Clin Psychol, 1976, 32(1), 149-153.

57. Gellhorn, E., and G.N. Louffburrow. Emotions and emotional disorders: Neurophysiological study. New York: Harper and Row, 1963.

58. Gellhorn, E. Conditioning, sensations, and the ergotrophic-trophotropic balance. Cond Reflex, 1968, 3, 34-44.

59. Gellhorn, E. Central nervous system tuning and its implications for neuropsychiatry. J Nerv Ment Dis, 1968, 147, 148-162.

60. Gellhorn, E. Further studies on the physiology and pathophysiology of the tuning of the central nervous system. Psychosomatics, 1969, 10, 94-104.

61. Wilder, J. The law of initial values in neurology and psychiatry. J Nerv Ment Dis, 1957, 13, 73-86.

62. Wolff, C.T., S.B. Friedman, M.A. Hofer, et al. Relationship between psychological defenses and mean urinary 17-OHCS excretion rates: I. A predictive study of parents of fatally ill children. Psychosom Med, 1964, 26, 576-591.

63. Bourne, P.G., R.M. Rose, and J.W. Mason. Urinary 17-OHCS levels: Data on seven helicopter ambulance medics in combat. Arch Gen Psychiatry, 1967, 17, 104.

64. Lacey, J.I. Individual differences in somatic response patterns. J Comp Physiol Psychol, 1950, 43, 338-350.

65. Lacey, J.I., and Ruth Van Lehn. Differential emphases in somatic response to stress. Psychosom Med, 1952, 14, 71-81.

66. Lacey, J.I., D.E. Bateman, and Ruth Van Lehn. Automatic response specificity and Rorschach color responses. Psychosom Med, 1952, 14, 256-260.

67. Lacey, J.I., D.E. Bateman, and Ruth Van Lehn. Autonomic response specificity: An experimental study. Psychosom Med, 1953, 15, 8-21.

68. Lacey, J.I., and B.C. Lacey. Verification and extension of the principle of autonomic response stereotypy. Am J Psychol, 1958, 71, 50-73.

69. Lacey, J.I., and B.C. Lacey. The law of initial value in the longitudinal study of autonomic responses and response patterns over a four-year interval. Ann NY Acad Sci, 1962, 98, 1257-1290.

70. Ax, A.F. The physiological differentiation between fear and anger in humans. Psychosom Med, 1953, 15, 433-442.

71. Funkenstein, D.H. The physiology of fear and anger. Sci Am, 1952, 192, 74.

72. Schacter, J. Pain, fear, and anger in hypertensives and normotensives. Psychosom Med, 1957, 19, 17-29.

73. Averill, J.R. Autonomic response patterns during sadness and mirth. Psychophysiology, 1969, 5, 399-414.

74. Masters, W.E., and V.E. Johnson. Human Sexual Response. Boston: Little, Brown, 1966.

75. Malhotra, M.S., and L. Mathew. Effect of prolonged stay at altitude (4000 m) on autonomic balance. Aerospace Med, 1974, 869-872.

76. Sersen, E.A., J. Clausen, and A. Lidsky. Autonomic specificity and stereotype revisited. Psychophysiology, 1978, 15(1), 60-67.

77. Block, J.D. Covariance analysis scale transformation in statistical treatment of non-additive units. Percept Mot Skills, 1964, 19, 755-761.

78. Benjamin, L.S. Facts and artifacts in using analysis of covariance to "undo" the law of initial values. Psychophysiology, 1967, 4, 187-202.

79. Sterman, M.B., T.L. Clemens, and M.A. Wenger. Electroencephalogram response to cold pressor stimulation. Psychophysiology, 1966, 2(4), 327-337.

80. Shipman, W.G., H.A. Heath, and D. Oken. Response specificity among muscular and autonomic variables. Arch Gen Psychiatry, 1970, 23, 369-374.

81. Zuckerman, Marvin, Harold Persky, and G.C. Curtis. Relationships among anxiety, depression, hostility, and autonomic variables. J Nerv Ment Dis, 1968, 146(6), 481-487.

82. Kelly, Desmond, C.C. Brown, and J.W. Schaffer. A comparison of physiological and psychological measurements on anxious patients and normal controls. Psychophysiology, 1970, 6(4), 429-441.

83. Surwit, R.S., R.N. Pilon, and C.H. Fenton. Behavioral treatment of Raynaud's disease. J Behav Med, 1978, 1(3), 323-335.

84. Taub, E., and C.F. Stroebel. Biofeedback in the treatment of vasconstrictive syndrome. Biofeedback Self Regul, 1978, 3(4), 363-373.

85. Jacobson, A.M., T.C. Manschreck, and E. Silverberg. Behavioral treatment for Raynaud's disease: A comparative study with long-term follow-up. Am J Psychiatry, 1979, 136(6), 844-846.

86. Jessup, B.A., R.W. Neufield, and H. Merskey. Biofeedback therapy for headaches and other pain: An evaluative review. Pain, 1979, 7(3), 225-270.

87. Prima, A., A. Agnoli, and A. Tamburello. A review of the applications of biofeedback to migraine and tension headaches. Acta Neurol, 1979, 34(6), 510-521.

88. Jankel, W.R. Bell palsy: Muscle reeducation by electromyography feedback. Arch Phys Med Rehabil, 1978, 59(5), 240-242.

89. Fernando, C.K., and J.V. Basmajian. Biofeedback in physical medicine and rehabilitation. Biofeedback Self Regul, 1978, 3(4), 435-455.

90. Burdick, J.A. A review of heart-rate variability and evaluation. Percept Mot Skills, 1978, 47(1), 95-105.

91. Burdick, J.A. Heart rate, tonic cardiac variability and evaluation. J Psychosom Res, 1978, 22(2), 69-77.

92. Burdick, J.A. Cardiac activity and attitude. J Pers Soc Psychol, 1972, 22, 80-86.

93. Burdick, J.A. Measurements of variability. J Gen Psychol, 1972, 86, 201-206.

94. Burdick, J.A. Heart rate variability (CVT); concurrent validity and test-retest reliability. Percept Mot Skills, 1968, 26, 1001-1003.

95. Light, K.C., and P.A. Obrist. Cardiovascular response to stress: Effects of opportunity to avoid shock experience, and performance feedback. Psychophysiology, 1980, 17(3), 243-252.

96. Obrist, P.A., K.C. Light, J.A. McCubbin, et al. Pulse transit time: Relationship to blood pressure and myocardial performance. Psychophysiology, 1979, 16(3), 292-301.

97. Obrist, P.A., K.C. Light, A.W. Langer, et al. Behavioral-cardiac interactions: The psychosomatic hypothesis. J Psychosom Res, 1978, 22(4), 301-325.

98. Obrist, P.A., C.J. Gaebelein, E.S. Teller, et al. The relationship among heart rate, carotid dP/dt, and blood pressure in humans as a function of the type of stress. Psychophysiology, 1978, 15(2), 102-115.

99. Obrist, P.A. The cardiovascular behavior interaction as it appears today. Psychophysiology, 1976, 13, 95-107.

100. Obrist, P.A., et al. Sympathetic influences on cardiac rate and contractility during acute stress in humans. Psychophysiology, 1974, 11(4), 405-427.

101. Lovallo, William, and A.R. Zeiner. Some factors influencing the vasomotor response to cold pressor stimulation. Psychophysiology, 1975, 12(5), 499-505.

102. Lovallo, W. The cold pressor test, an autonomic function: A review and integration. Psychophysiology, 1975, 12(3), 268-282.

103. Lovallo, W., A.A. Parson, and F.A. Holloway. Autonomic arousal in normal, alcoholic, and brain-damaged subjects as measured by the plethysmograph response to cold pressor stimulation. Psychophysiology, 1973, 10, 166-176.

104. von Neumann, J., R.H. Kent, H.R. Bellinson, et al. The mean square successive difference. Ann Math Stat, 1941, 12, 153-162.

105. von Neumann, J. Distribution of the ration of the mean square successive difference to the variance. Ann Math Stat, 1941, 12, 367-395.

106. Hinman, A.T., B.T. Engel, and A.F. Bickford. Portable blood pressure recorder: Accuracy and preliminary use in evaluating daily variations in pressure. Am Heart J, 1962, 63, 663-668.

107. Lategola, M.T., H. Harrison, and C. Barnard. Continuous measurement of human blood pressure transients without puncture. Aerospace Med, 1966, 37, 228-233.

108. Tursky, B. The indirect recording of human blood pressure. In P.A. Obrist et al. (eds.). Cardiovascular Psychophysiology: Current Issues in Response Mechanisms, Biofeedback and Methodology. Chicago: Aldine Atherton, 1974, 93-105.

109. Obrist, P.A., and J.S. Hutcheson. An automated non-invasive blood pressure monitor: Principle of operation and reliability. Unpublished manuscript, 1978. (Available on request from the first author.)

110. Tursky, B., D. Shapiro, and G.E. Schwartz. Automated constant cuff-pressure system to measure average systolic and diastolic blood pressure in man. IEEE Trans Biomed Eng, 1972, 19, 271-276.

111. Williams, J.G.L., and B. Williams. Arterial pulse wave velocity as a psychophysiological measure. Psychosom Med, 1965, 27, 408-414.

112. Thomas, J.G. Continuous pulse wave velocity recording for indirectly monitoring blood pressure in man. Med Electron Biol Eng, 1965, 3, 331-332.

113. Gribbin, B., A. Steptoe, and P. Sleight. Pulse wave velocity as a measure of blood pressure change. Psychophysiology, 1976, 13, 86-90.

114. Steptoe, A., H. Smulyan, and B. Gribbin. Pulse wave velocity and blood pressure change: Calibration and applications. Psychophysiology, 1976, 13(5), 488-493.

115. Obrist, P.A. The relationship between pulse transit time and systolic blood pressure during rest and stress. Unpublished technical document for Cyborg. Personal communication, 1979

116. Sewall, H., and E. Sanford. Plethysmographic studies of the human vaso-motor mechanisms when excited by electrical stimulation. J Physiol, 1890, London, 11, 179-207.

117. Brown, C. Instruments in Psychophysiology. In N.S. Greenfield and R.A. Sternbach (eds.). Handbook of Psychophysiology. New York: Holt Rinehart, 1972, 159-196.

118. Burch, G.E. Digital Plethysmography. New York: Grune and Stratton, 1954.

119. Turpin, G., and D.A. Siddle. Cardiac and forearm plethysmographic responses to high intensity auditory stimulation. Biol Psychol, 1978, 6(4), 257-281.

120. Henson, C., H.B. Rubin, and D.E. Henson. Women's sexual arousal concurrently assessed by three genital measures. Arch Sex Behav, 1979, 8(6), 459-469.

121. Whitney, R.J. The electrical strain gauge method for measurement of peripheral circulation in man. In G.E. Wolstenholme (ed.). Peripheral Circulation in Man. Boston: Little, Brown, 1954.

122. Freund, K., F. Sedlacek, and K. Knob. A simple transduce for mechanical plethysmography of the male genital. J Exp Anal Behav, 1965, 8, 169-170.

123. Barry, J.M., and C.V. Hodges. Impotence: A diagnostic approach. J Urol, 1978, 119(4), 575-578.

124. Brown, C.C., D.B. Giddon, and E.D. Dean. Techniques of plethysmography. Psychophysiology, 1965, 1, 253-266.

125. Fredrikson, M., and A. Ohman. Cardiovascular and electrodermal responses conditioned to fear-relevant stimuli. Psychophysiology, 1979, 16(1), 1-7.

126. Hinton, J.W., and Brian Craske. Differential effects of test stress on the heart rates of extroverts and introverts. Biol Psychol, 1977, 5(1), 23-28.

127. Bloom, L.J., and G.M. Trautt. Finger pulse volume as a measure of anxiety: further evaluation. Psychophysiology, 1977, 14, 541-544.

128. Lerro, F.A., M.M. Hurnyak, and C. Patterson. Successful use of thermal biofeedback in severe adult asthma. Am J Psychiatry, 1980, 137(6), 735-736.

129. Lieberman, J.S. Instrumental methods in the study of vascular disease. Am Heart J, 1980, 99(4), 517-527.

130. Barnes, R.W. Noninvasive diagnostic techniques in peripheral vascular disease. Am Heart J, 1979, 97(2), 241-258.

131. Edelberg, R. Electrical properties of the skin. In C.C. Brown (ed.). Methods in Psychophysiology. Baltimore: Williams and Wilkins, 1967.

132. Wilcott, R.C. Arousal sweating and electrodermal phenomena. Psychol Bull, 1967, 67, 58-72.

133. Edelberg, R. Electrical activity of the skin: Its measurement and uses in psychophysiology. In N.S. Greenfield and R.A. Sternbach (eds.). Handbook of Psychophysiology. New York: Holt Rinehart, 1972, 367-418.

134. Goldstein, I.B. Electromyography: A measure of skeletal muscle response. In N.S. Greenfield and R.A. Sternbach (eds.). Handbook of Psychophysiology, New York: Holt Rinehart, 1972, 329-366.

135. Lashley, K.A. Reflex secretion of human parotid gland. J Exp Psychol, 1916, 1, 461-493.

136. Bogdonoff, M.D., M.M. Bogdonoff, and S.G. Wolf. Studies of salivar function in man: Variations in secretory rate as part of the individual's adaptive pattern. J Psychosom Res, 1961, 5, 170-174.

137. Corcoran, D.W.J. The relation between introversion and salivation. Am J Psychol, 1964, 77, 298-300.

138. Eysenck, S.B.G., and H.J. Eysenck. Salivary response to lemon juice as a measure of introversion. Percept Mot Skills, 1967, 24, 1047-1053.

139. Bolwig, T.G., and O.J. Rafaelsen. Salivation in affective disorders. Psychosom Med, 1972, 2, 232-238.

140. Peck, R.E. The SHP test - an aid in the detection and measurement of depression. Arch Gen Psychiatry, 1959, 1, 35-40.

141. Brown, C.C. The parotid puzzle: A review of the literature on human salivation and its application to psychophysiology. Psychophysiology, 1970, 7, 66-85.

142. White, K.D. Salivation: A review and experimental investigation of major techniques. Psychophysiology, 1977, 14(2), 203-212.

143. Greenfield, N.S., and R.A. Sternbach (eds.). Handbook of Psychophysiology, 1972, New York: Holt Rinehart.

144. Berman, M.L., and J.A. Pettitt. Urinary excretion of 3-methoxy-4-hydroxymandelic acid after several stress situations. J Lab Clin Med, 1961, 57, 126.

145. Taggart, P., and M. Carruthers. Endogenous hyperlipidaemia induced by emotional stress of racing driving. Lancet, 1971, 1, 363.

146. Gerdes, E.P. Autonomic arousal as a cognitive cue in stressful situations. J Pers, 1979, 47(4), 677-711.

147. Rahe, R.H., et al. Serum lactic acid variability in subjects experiencing stressful training. Dis Nerv Syst, 1972, 33(6), 403-408.

148. Stokols, Daniel, et al. Traffic congestion, Type A behavior, and stress. J Appl Psychol, 1978, 63(4), 467-480.

149. Bogdonoff, M., H. Estes, W. Harlan, et al. Metabolic and cardiovascular changes during a state of acute central nervous system arousal. J Clin Endocrinol, 1960, 20(10), 1333-1340.

150. Jones, M.T., P.K. Bridges, and D. Leak. Relationship between the cardiovascular and sympathetic response to the psychological stress of an examination. Clin Sci, 1968, 35, 73.

151. Bircher, M., J. Kohl, B. Nigg, et al. The microvibrations of the body, an index for examination stress. Eur J Appl Physiol, 1978, 39(2), 99-109.

152. Simpson, M.T., D.A. Olewine, C.D. Jenkins, et al. Exercise-induced catecholamines and platelet aggregation in the coronary-prone behavior pattern. Psychosom Med, 1974, 36(6), 476-487.

153. Averill, J.R. Personal control over aversive stimuli and its relationship to stress. Psychol Bull, 1973, 80(4), 286-303.

154. Weidner, G., and K.A. Matthews. Reported physical symptoms elicited by unpredictable events and the type A coronary-prone behavior pattern. J Pers Soc Psychol, 1978, 36(11), 1213-1220.

155. Moray, N. (ed.). Mental Workload, Its Theory and Measurement. New York: Plenum Press, 1979.

156. Wierwille, W.W. Physiological measures of aircrew mental workload. Hum Factors, 1979, 21(5), 575-593.

157. Krantz, D.S., D.C. Glass, and M.L. Synder. Helplessness, stress level, and the coronary-prone behavior pattern. J Exp Soc Psychol, 1974, 10, 284-300.

158. Engel, B.T., and A.F. Bickford. Response-specificity: Stimulus-response and individual-response specificity in essential hypertensives. Arch Gen Psychiatry, 1961, 5, 478-489.

159. Engel, B.T. Stimulus-response and individual-response specificity. Arch Gen Psychiatry, 1960, 2, 305-313.

160. Brod, J., V.Z. Fend, and J.J. Hejl. Circulatory changes underlying blood pressure elevation during acute emotional stress (mental arithmetic) in normotensive and hypertensive subjects. Clin Sci Mol Med, 1959, 18, 269-279.

161. Bonelli, J., H. Hortnagl, J. Brucke, et al. Effect of calculation stress on hemodynamics and plasma catecholamines before and after beta-blockade with propranolol (Inderal) and mepindolol sulfate (Corindolan). Eur J Clin Pharmacol, 1979, 15(1), 1-8.

162. Collins, A., and M. Frankenhaeuser. Stress responses in male and female engineering students. J Hum Stress, 1978, 4, 43-48.

163. Averill, J., and M. Rosenn. Vigilant and non-vigilant coping strategies and psychophysiological stress reactions during the anticipation of electric shock. J Pers Soc Psychol, 1972, 23, 128-141.

164. DeBusk, R.F., C.B. Taylor, and W.S. Agras. Comparison of treadmill exercise testing and psychologic stress testing soon after myocardial infarction. Am J Cardiol, 1979, 43(5), 907-912.

165. Frankenhaeuser, Marianne, Elizabeth Dunne, and Ulf Lundberg. Sex differences in sympathetic adrenal medullary reactions induced by different stressors. Psychopharmacology, 1976, 47(1), 1-5.

166. Coleman, D.J., and G.E. Schwartz. Meditation as an intervention in stress reactivity. J Consult Clin Psychol, 1976, 44, 456-466.

167. Huchingson, R.D. Effects of time constraint on closed-loop tracking performance and change in heart rate. Percept Mot Skills, 1973, 36(3-2), 1195-1198.

168. Klorman, R., A.R. Wiesenfeld, and M.L. Austin. Autonomic responses to affective visual stimuli. Psychophysiology, 1975, 12, 553-560.

169. Bogdonoff, M.Q., J.J. Combs, G.D.N. Bryant, et al. Cardiovascular responses in experimentally induced alterations of affect. Circulation, 1959, 20, 353-359.

170. Petry, H.M., and Otello Desiderato. Changes in heart rate, muscle activity, and anxiety level following shock threat. Psychophysiology, 1978, 15(5), 398-402.

171. Hein, P.L., I.D. Sanford, S.I. Cohen, et al. Perceptual mode and cardiac conditioning. Psychophysiology, 1966, 3, 101-107.

172. Van Egeren, L.F., J.L. Abelson, and D.W. Thorton. Cardiovascular consequences of expressing anger in a mutually dependent relationship. J Psychosom Res, 1978, 22(6), 537-548.

173. Cantor, J.R., D. Zillman, and K.D. Day. Relationship between cardiorespiratory fitness and physiological responses to films. Percept Mot Skills, 1978, 46(3-2), 1123-1130.

174. Soskek, A.J. Effects of electrodermal lability and payoff instructions on vigilance performance. Psychophysiology, 1978, 15(6), 561-568.

175. Ohman, A., M. Fredrikson, and K. Hugdahl. Orienting and defensive responding in the electrodermal system: Palmar-dorsal differences and recovery rate during conditioning to potentially phobic stimuli. Psychophysiology, 1978, 15(2), 93-101.

176. Bundy, R.S., and S.M. Mangan. Electrodermal indices of stress and cognition: possible hydration artifacts. Psychophysiology, 1979, 16(1), 30-33.

177. Cacioppo, J.T., and C.A. Sandman. Physiological differentiation of sensory and cognitive tasks as a function of warning, processing demands, and reported unpleasantness. Biol Psychol, 1978, 6(3), 181-192.

178. Manuck, S.B., A.H. Harvey, Stephanie Lechleiter, et al. Effects of coping on blood pressure responses to threat of aversive stimulation. Psychophysiology, 1978, 15(6), 544-549.

179. Sandman, C.A. Physiological responses during escape and non-escape from stress in field independent and field dependent subjects. Biol Psychol, 1975, 2(3), 205-216.

180. Almy, T.P. The stress interview: Unfinished business. J Hum Stress, 1978, 4(4), 3-8.

181. Brown, W.A., and G. Heninger. Cortisol, growth hormone, free fatty acids, and experimentally evoked affective arousal. Am J Psychiatry, 1975, 132, 1172-1176.

182. Crow, L.T., and C. Ball. Alcohol state-dependency and autonomic reactivity. Psychophysiology, 1975, 12(6), 702-706.

183. DeGood, D.E. Cognitive control factors in vascular stress responses. Psychophysiology, 1975, 12(4), 399-401.

184. Fine, B.J., and D.R. Sweeney. Personality traits, and situational factors, and catecholamine excretion. J Exp Res Pers, 1968, 3(1), 15-27.

185. Johansson, Gunn. Subjective well-being and temporal patterns of sympathetic-adrenal medullary activity. Biol Psychol, 1976, 4(3), 157-172.

186. Johansson, Gunn, and Marianne Frankenhaeuser. Temporal factors in sympatho-adrenomedullary activity following acute behavioral activation. Reports from the Psychological Lab., U. Stockholm, 1972, 353, 1-11.

187. Lintel, A.G., III. Physiological anxiety responses in transcendental meditators and nonmeditators. Percept Mot Skills, 1980, 50(1), 295-300.

188. Lobstein, T., B. Webb, and O. Edholm. Orienting responses and locus of control. Br J Soc Clin Psychol, 1979, 18(1), 13-19.

189. Notarius, C.I., and R.W. Levenson. Expressive tendencies and physiological response to stress. J Pers Soc Psychol, 1979, 37(7), 1204-1210.

190. Robertson, D., G.A. Johnson, R.M. Robertson, et al. Comparative assessment of stimuli that release neuronal and adrenomedullary catecholamines in man. Circulation, 1979, 59(4), 637.

191. Terry, R.A. Autonomic balance and temperament. J Comp Physiol Psychol, 1953, 46, 454-460.

192. Averill, J.R., Erhard Olbrich, and R.S. Lazarus. Personality correlates of differential responsiveness to direct and vicarious threat: A failure to replicate previous findings. J Pers Soc Psychol, 1972, 21(1), 25-29.

193. Badian, M., E. Appel, D. Palm, et al. Standardized mental stress in healthy volunteers induced by delayed auditory feedback (DAF). Eur J Clin Pharmacol, 1979, 16(3), 171-176.

194. Hafner, R.J. Relationships between personality and autonomic nervous reactions to stress. J Psychosom Res, 1974, 18(3), 181-185.

195. Curtis, R.C., A. Mariano, and W.D. Gersh. Probabilities of an aversive event and physiological correlates of stress. Psychol Rep, 1979, 44(1), 247-256.

196. Hare, R.D., J. Frazelle, and D.N. Cox. Psychopathy and physiological responses to threat of an aversive stimulus. Psychophysiology, 1978, 15(2), 165-172.

197. Berkeley, A.W. Level of aspiration in relation to adrenal cortical activity and the concept of stress. J Comp Physiol Psychol, 1952, 45, 443-449.

198. Houston, B.K. Control over stress, locus of control and response to stress. J Pers Soc Psychol, 1972, 21, 249-255.

199. Nestel, P.J. Blood pressure and catecholamine excretion after mental stress in labile hypertension. Lancet, 1969, 1, 692-694.

200. Levi, L. (ed.). Stress and distress in response to psychosocial stimuli. Laboratory and real life studies on sympathoadrenomedullary and related reactions. Acta Med Scand, 1972, Suppl. 528.

201. Lazarus, R.S., J.C. Speisman, and A.M. Mordkoff. The relationship between autonomic indicators of psychological stress: heart rate and skin conductance. Psychosom Med, 1963, 25, 19.

202. Taggart, P., P. Parkinson, and M. Carruthers. Cardiac responses to thermal, physical and emotional stress. Br Med J, 1972, 3, 71.

203. Matthews, K.A., and B.I. Brunson. Allocation of attention and the Type A coronary-prone behavior pattern. J Pers Soc Psychol, 1979, 37(11), 2081-2090.

204. Petersen, C.C., A. Ravindran, et al. Modeling and analysis in support of the HEART (Health Evaluation And Risk Tabulation) program. USAF School of Aerospace Medicine Technical Report No. 0153-53-1287, 1979.

TABLE 1. PSYCHOPHYSIOLOGICAL VARIABLES USED IN CITED RESEARCH STUDIES

Variables	Reference Citations																			
	4	23	25	26	27	28	29	30	35	42	43	44	47	48	49	50	52	53	54	56
Heart rate	+			+	+	+	+		+	+	+	+	+	+	+	+	+	+	+	+
Blood pressure	b		b	b	b	b	b	b	b	s	b	b	b	b	b	b			s	b
Transit time/PWV																				
Blood flow change									o		t	t,v	t	t	v	t,v			t	
Respiration										+	+	+	+	+	+	+	+	+		+
Electrodermal				p	p					r	r	r	r	r	r	r		r	r	r
Muscle tension												f,a								
Others	p	p								s,d	s,d	s,d	s,d	s,d		s,d				s,d

Blood pressure: s = systolic; d = diastolic; b = both

Blood flow change: v = volume/girth; o = optical; t = temperature

Electrodermal: p = potential (endosomatic); r = resistance/conductance (DC)

Muscle tension: *f = frontalis; a = forearm*

Others: s = salivation; d = dermographia; p = serum epinephrine, norepinephrine, cortisol

TABLE 1. (Continued)

Variables	62	63	64	65	66	67	68	70	72	75	76	79	80	81	82	91	92	95	96	98
	Reference Citations																			
Heart rate			+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+
Blood pressure			b	b			b		b	b	s	b	b					b	b	b
Transit time/PWV																		+	+	+
Blood flow change								t	t	t		v			v,o					
Respiration								+	+	+	+	+		+						
Electrodermal			r	r	r	r	r	r	r		r	r	r	r	r					
Muscle tension									f				+		a					
Others	u	u							s,e		e									

Blood pressure: s = systolic; d = diastolic; b = both

Blood *flow change*: v = *volume/girth*; o = *optical*; t = *temperature*

Electrodermal: r = resistance/conductance (DC)

Muscle tension: f = frontalis; a = forearm; + = other

Others: s = salivation; u = urine epinephrine, norepinephrine, cortisol;
e = electroencephalogram

TABLE 1. (Continued)

Variables	99	100	101	103	113	114	115	119	120	125	126	127	136	144	146	158	159	160	161	162
	Reference Citations																			
Heart rate	+	+						+		+	+	+			+	+	+		+	+
Blood pressure	b	b			b	c	c								b	b	b	b	b	
Transit time/PWV	+	+	+		+	+	+													
Blood flow change			v	v				v	v,o	o	o	o				t	t			
Respiration																+	+			
Electrodermal										r						r	r			
Muscle tension		+																		
Others													s	u					p	u

Blood pressure: b = both systolic and diastolic; c = catheter

Blood flow change: v = volume/girth; o = optical; t = temperature

Electrodermal: r = resistance/conductance (DC)

Others: s = salivation; u = urine epinephrine, norepinephrine, cortisol;
p = serum epinephrine, norepinephrine, cortisol

TABLE 1. (Continued)

Variables	163	164	165	166	167	168	169	170	171	172	173	174	175	176	177	178	179	181	182	183
	Reference Citations																			
Heart rate	+	+	+	+	+	+	+	+	+	+	+				+		+		+	
Blood pressure		b					c			b	s					b				b
Transit time/PWV										+										
Blood flow change										o	t								o	
Respiration	+					+	+			+		+	+		+		+		+	
Electrodermal	r			r		r						r	r	r	p		p,r		r	
Muscle tension								a												
Others			p															p	e	

Blood pressure: b = both systolic and diastolic; s = systolic; c = catheter

Blood flow change: o = optical; t = temperature

Electrodermal: r = resistance/conductance (DC); p = potential (endosomatic)

Muscle tension: a = forearm

Others: p = serum epinephrine, norepinephrine, cortisol;
u = urine epinephrine, norepinephrine, cortisol

TABLE 1. (Continued)

Variables	Reference Citations																		
	184	185	186	187	188	189	190	191	192	193	194	195	196	197	198	199	200	201	202
Heart rate			+		+	+		+	+	+	+	+	+		+			+	+
Blood pressure								b		b						b			
Transit time/PWV																			
Blood flow change											o		o						
Respiration						+							+						
Electrodermal				r	r	r		p,r	r			r	r					r	
Muscle tension																			
Others	u	u	u				p,u			p	s			u		u	u		

Blood pressure: b = both systolic and diastolic

Blood flow change: o = optical

Electrodermal: r = resistance/conductance (DC); p = potential (endosomatic)

Others: u = urine epinephrine, norepinephrine, cortisol; p = serum epinephrine, norepinephrine, cortisol; s = salivation

TABLE 2. LABORATORY TASKS/SITUATIONS USED IN EACH RESEARCH STUDY CITED

	Reference Citations																		
	4	23	24	25	26	27	28	29	30	32	33	35	49	53	54	64	65	66	67
Aversive Stimuli/Situations																			
Cold pressor test												x					x	x	x
Electric shock																			
Shock-conditioned stimuli																			
Threat of shock/aversive stim.			x																
False emergency																			
Films/slides (autopsy, etc)															x				
Harassment																			
Noise (loud or distracting)	x	x												x					
Temperature (hot or cold)															x				
Venipuncture		x																	
Cognitive Tasks																			
Arithmetic problems	x									x		x						x	x
Conflict (e.g., Stroop, aud fdbk)																			
Interviews				x					x			x							
Memorization																			
Verbal/concept problems		x		x		x	x	x							x				
Word associations																x		x	x
Psychomotor																			
Computer game						x													
Maze or tracing task																			
Reaction time					x	x													
Vigilance, DRL, time estim										x	x								
Other																			
Hyperventilation																		x	x
Balloon inflation																			
Isometrics, Valsalva, exercise																			
Music, poem, bland films															x				
Pornographic/erotic stimuli													x						
Unsolvable prob/false stds		x					x	x											

TABLE 2. (Continued)

	Reference Citations																		
	68	70	72	75	76	79	80	81	82	91	95	96	98	99	100	101	102	103	114
Aversive Stimuli/Situations																			
Cold pressor test	x		x	x	x	x		x				x	x	x		x	x	x	
Electric shock		x	x								x	x	x	x	x				
Shock-conditioned stimuli																			
Threat of shock/aversive stim.																			
False emergency		x	x				x												
Films/slides (autopsy, etc)																			
Harassment		x	x						x										
Noise (loud or distracting)					x		x						x	x					
Temperature (hot or cold)																			
Venipuncture																			
Cognitive Tasks																			
Arithmetic problems	x				x				x										x
Conflict (e.g., Stroop, aud fdbk)																			
Interviews																			
Memorization																			
Verbal/concept problems																			
Word associations	x																		
Psychomotor																			
Computer game																			
Maze or tracing task																			
Reaction time											x	x		x	x				
Vigilance, DRL, time estim																			
Other																			
Hyperventilation																			
Balloon inflation																			
Isometrics, Valsalva, exercise																			x
Music, poem, bland films		x	x		x														
Pornographic/erotic stimuli										x		x	x	x					
Unsolvable prob/false stds																			

TABLE 2. (Continued)

	Reference Citations																		
	115	119	125	126	127	136	144	146	157	158	159	160	161	162	163	164	165	166	167
Aversive Stimuli/Situations																			
Cold pressor test	x									x	x								
Electric shock			x												x				
Shock-conditioned stimuli			x																
Threat of shock/aversive stim.					x	x		x											
False emergency																			
Films/slides (autopsy, etc)																x		x	
Harassment		x																	
Noise (loud or distracting)		x							x	x	x								
Temperature (hot or cold)							x												
Venipuncture																	x		
Cognitive Tasks																			
Arithmetic problems	x	x		x						x	x	x	x						
Conflict (e.g., Stroop, aud fdbk)														x			x		
Interviews																x			
Memorization																			
Verbal/concept problems										x	x					x			
Word associations																			
Psychomotor																			
Computer game																			
Maze or tracing task																			x
Reaction time																			
Vigilance, DRL, time estim															x				
Other																			
Hyperventilation																			
Balloon inflation																			
Isometrics, Valsalva, exercise										x	x					x			
Music, poem, bland films																			
Pornographic/erotic stimuli	x														x				
Unsolvable prob/false stds		x		x															

TABLE 2. (Continued)

	Reference Citations																		
	168	169	170	171	172	173	174	175	176	177	178	179	180	181	182	183	184	185	186
Aversive Stimuli/Situations																			
Cold pressor test																			
Electric shock		x	x					x								x			
Shock-conditioned stimuli				x				x											
Threat of shock/aversive stim.									x										
False emergency		x																	
Films/slides (autopsy, etc)	x					x				x		x							
Harassment					x														
Noise (loud or distracting)											x								
Temperature (hot or cold)																	x		
Venipuncture																			
Cognitive Tasks																			
Arithmetic problems										x								x	
Conflict (e.g., Stroop, aud fdbk)																		x	
Interviews									x				x						
Memorization										x									
Verbal/concept problems					x					x					x				
Word associations															x				
Psychomotor																			
Computer game																			
Maze or tracing task									x						x				
Reaction time								x											x
Vigilance, DRL, time estim							x												
Other																			
Hyperventilation																			
Balloon inflation									x										
Isometrics, Valsalva, exercise																			
Music, poem, bland films	x							x						x					
Pornographic/erotic stimuli						x								x					
Unsolvable prob/false stds																			

TABLE 2. (Continued)

	Reference Citations																
	187	188	189	190	191	192	193	194	195	196	197	198	199	200	201	202	203
Aversive Stimuli/Situations																	
Cold pressor test				x													
Electric shock	x								x			x					
Shock-conditioned stimuli																	
Threat of shock/aversive stim.			x			x											
False emergency																	
Films/slides (autopsy, etc)														x	x		
Harassment																	
Noise (loud or distracting)		x						x		x							
Temperature (hot or cold)																x	
Venipuncture																	
Cognitive Tasks																	
Arithmetic problems					x			x									
Conflict (e.g., Stroop, aud fdbk)							x										x
Interviews																	
Memorization												x					
Verbal/concept problems									x				x				
Word associations																	
Psychomotor																	
Computer game																	
Maze or tracing task											x						
Reaction time																	x
Vigilance, DRL, time estim																	
Other																	
Hyperventilation																	
Balloon inflation																	
Isometrics, Valsalva, exercise				x													
Music, poem, bland films					x										x		
Pornographic/erotic stimuli														x			
Unsolvable prob/false stds											x						

ATE

LMED

-8